Delgocitinib ointment, a topical Janus kinase inhibitor, in adult patients with moderate to severe atopic dermatitis: A phase 3, randomized, double-blind, vehicle-controlled study and an open-label, long-term extension study

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Background: Previous studies showed the potential effectiveness of delgocitinib ointment, a novel topical Janus kinase inhibitor, in atopic dermatitis (AD).

Objective: This study aimed to evaluate the efficacy and safety of delgocitinib 0.5% ointment.

Methods: In part 1, a 4-week double-blind period, Japanese patients aged 16 years or older with moderate or severe AD were randomly assigned in a 2:1 ratio to delgocitinib 0.5% ointment or vehicle ointment. Eligible patients entered part 2, a 24-week extension period, to receive delgocitinib 0.5% ointment.

Results: At the end of treatment in part 1, the least-squares mean percent changes from baseline in the modified Eczema Area and Severity Index score, the primary efficacy endpoint, were significantly greater in the delgocitinib group than in the vehicle group (-44.3% vs 1.7%, \( P < .001 \)). The improvement in modified Eczema Area and Severity Index score was maintained in part 2. Most adverse events were mild and unrelated to delgocitinib across the study periods.

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The results of part 1 of the study were presented at the 2019 American Academy of Dermatology Annual Meeting in Washington, DC, on March 1-2, 2019.

IRB approval status: Study-related documents, including the study protocol and informed consent forms, were reviewed by the Sugiura Clinic IRB on April 4, 2017, Atago Dermatology Clinic IRB on April 28, 2017, Osaka University Hospital IRB on March 28, 2017, Tokyo Teishin Hospital IRB on April 19, 2017, and Jichi Medical University Hospital IRB on April 28, 2017. The conduct of the study was approved for all the study sites.

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Limitations: Only Japanese patients were included. The vehicle-controlled period lasted only 4 weeks. In part 2, topical corticosteroids were allowed for the treatment of worsening of AD.

Conclusion: Delgocitinib ointment was effective and well tolerated in Japanese adult patients with moderate to severe AD for up to 28 weeks. (J Am Acad Dermatol 2020;82:823-31.)

Key words: Atopic dermatitis; delgocitinib; eczema; inflammation; JAK inhibitor; Janus kinase; JTE-052; ointment; pruritus; QOL; skin barrier; topical therapy.

Atopic dermatitis (AD) is a pruritic, eczematous dermatitis, and its symptoms chronically fluctuate with remissions and relapses.\(^1,2\) Topical corticosteroids and calcineurin inhibitors form the mainstay of controlling skin inflammation of AD. These drugs, however, present safety concerns, such as skin atrophy and telangiectasia for topical corticosteroids and skin irritation symptoms for tacrolimus ointment.\(^1\) Therefore, novel topical treatment options with a better efficacy-safety profile are still needed.

Features of AD can be explained by immunologic abnormalities, skin barrier dysfunction, and pruritus.\(^3,5\) Immunologic abnormalities include the enhanced production of inflammatory cytokines.\(^6-10\) Skin barrier dysfunction is associated with a reduction in filaggrin production caused by mutations in the filaggrin genes and the overexpression of interleukin (IL) 4 and IL-13.\(^11-13\) Pruritus has been shown to be induced by IL-31.\(^15,16\) Additionally, recent reports indicate that AD is a highly heterogeneous disease, with various subtypes and phenotypes depending on the patients’ backgrounds, such as ethnicity and age, and is also characterized by the coexistence of abnormalities in cytokine production of T helper (Th) type 1, Th2, Th17, and Th22.\(^9,10,17-20\) In addition to targeting Th2 cytokines such as IL-4, IL-13, and IL-31, targeting other cytokine axes is a theoretically beneficial strategy for the treatment of AD.\(^9,10\) Taken together, the broad regulation of abnormal cytokine activities can be a potential target for novel treatment options in AD.

Various cytokines exert their biological effects via the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) pathway.\(^21,22\) Several JAK inhibitors are currently under development for the treatment of AD.\(^25,24\) Delgocitinib (formerly JTE-052) is a novel, small-molecule (molecular weight, 310.35) JAK inhibitor under development in Japan by Japan Tobacco and Torii Pharmaceutical.

Delgocitinib has inhibitory effects on JAK1, JAK2, JAK3, and tyrosine kinase 2.\(^25\) In preclinical studies, topical application of delgocitinib suppressed skin inflammation,\(^26\) improved skin barrier dysfunction,\(^27\) and suppressed pruritus induced by IL-31.\(^28\) These findings indicate that topical delgocitinib can be a novel drug for the treatment of AD.

Previous studies showed the potential effectiveness of delgocitinib ointment in Japanese adult patients with AD.\(^29,30\) In the present phase 3 study, we evaluated the efficacy and safety of delgocitinib 0.5% ointment in Japanese adult patients with moderate to severe AD over a 4-week double-blind period (part 1) and a 24-week extension period (part 2).

METHODS

Study design

Part 1 was a 4-week, randomized, double-blind, vehicle-controlled study. Patients were randomized in a 2:1 ratio to delgocitinib 0.5% ointment or vehicle ointment (Supplemental Fig 1; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1). A computer-generated randomization was performed with a dynamic allocation method, and the randomization was stratified by Investigator’s Global Assessment (IGA) score at baseline. After completion of part 1, patients could enter part 2, which was a 24-week, open-label extension study. When patients did not complete part 1 because of worsening of AD, they were discontinued from the study or entered into part 2 early at the investigators’ discretion. In part 2, all patients received delgocitinib 0.5% ointment.
This study was conducted at 24 medical institutions in Japan in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines. Study-related documents, including the study protocol and informed consent forms, were approved by the institutional review boards. Written informed consent was obtained from all patients. The study information is registered with Japan Pharmaceutical Information Center (JAPIC) Clinical Trials Information (www.clinicaltrials.jp), number JapicCTI-173554.

Patients

Japanese patients aged 16 years or older and diagnosed with AD according to the criteria of the Japanese Dermatological Association1 were enrolled. At initiation of part 1, patients were required to have a modified Eczema Area and Severity Index (mEASI) score of 10 or greater (mEASI score was calculated by excluding the head/neck region score from the EASI31 total score); an IGA score of 3 (moderate) or 4 (severe); and inflammatory eczema affecting 10% to 30% of the body surface area (BSA). Exclusion criteria are summarized in the Supplemental Appendix (available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

Study treatment

Patients were instructed to apply the ointment twice daily (maximum dose per application, 5 g) to the areas affected by inflammatory eczema, excluding dry skin areas and the scalp. In part 1, concomitant use of any therapy to the application areas was prohibited. However, in part 2, topical corticosteroids for the treatment of worsening of AD could be used at the investigators’ discretion. Other prohibited and permitted concomitant therapies are summarized in the Supplemental Appendix.

Study assessments

Efficacy assessments were based on the following: mEASI, EASI, IGA, face/neck IGA, pruritus numeric rating scale (NRS), percentage of BSA affected by AD, and Skindex-16,32,33 which are detailed in the Supplemental Appendix. In part 1, the primary efficacy endpoint was the percent change from baseline in the mEASI score at the end of treatment (EOT). Secondary efficacy endpoints included the changes or percent changes from baseline in the other parameters at EOT. Secondary endpoints also included the proportions of patients who achieved the following criteria at EOT: at least 50% improvement from baseline in the mEASI score (mEASI-50), at least 75% improvement from baseline in the mEASI score (mEASI-75), an IGA score of 0 (clear) or 1 (almost clear) with at least 2-point improvement from baseline, and a face/neck IGA score of 0 or 1 with at least 2-point improvement from baseline. Long-term efficacy was assessed with the mEASI, IGA, and pruritus NRS scores across parts 1 and 2. Safety assessments were based on symptoms, signs, vital signs, and laboratory test results. Plasma concentrations of delgocitinib were measured at selected visits, and the lower limit of quantification was 1.00 ng/mL.

Statistical analyses

The sample-size calculation was based on the results of a phase 2 study of delgocitinib ointment in adult patients with AD.30 The sample size (100 for delgocitinib 0.5%, 50 for the vehicle) would provide at least 90% power to detect a significant difference between delgocitinib 0.5% and vehicle groups in the primary efficacy endpoint, the percent change from baseline in the mEASI score at EOT, with a 1-sided test at the 2.5% significance level.

Primary analyses of efficacy, safety, and pharmacokinetics were performed on the population comprising randomized patients who underwent the respective study-specified assessments at least once after the start of study treatment. The EOT value for efficacy assessments was defined as the value at week 4, study discontinuation, or immediately before part 2. For long-term efficacy assessments, baseline (week 0) was defined as the first day of delgocitinib treatment (ie, the first day of part 2 for patients receiving the vehicle ointment in part 1).

The primary and secondary efficacy endpoints were analyzed with analysis of covariance, with the relevant baseline value as the covariate. The
least-squares mean change or percent change from baseline was calculated. For responder analyses of mEASI and IGA scores, odds ratios were calculated by using logistic regression, with the fixed effect for the treatment groups and the baseline value as the covariate. All statistical tests are 1-sided at the 2.5% significance level, and no multiplicity adjustment was performed.

RESULTS

Patients

A total of 158 patients were randomized in part 1 (Supplemental Fig 2; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1). Of 106 patients in the delgocitinib group, 98 (92.5%) completed part 1, and 8 (7.5%) entered part 2 early. Of 52 patients in the vehicle group, 29 (55.8%) completed part 1, 20 (38.5%) entered part 2 early, and 3 (5.8%) were discontinued from the study. A total of 154 patients entered part 2, and 138 (89.6%) patients completed part 2.

No apparent differences between treatment groups in part 1 were found in the demographic and baseline characteristics (Table I). Because of worsening of AD, topical corticosteroids were used at least once by 64 (41.6%) patients in part 2.

Table I. Patient demographics and baseline characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Vehicle ointment (n = 52)</th>
<th>Delgocitinib 0.5% ointment (n = 106)</th>
<th>Total (N = 158)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>32.3 (11.2)</td>
<td>31.4 (9.6)</td>
<td>31.7 (10.1)</td>
</tr>
<tr>
<td>Sex, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>34 (65.4)</td>
<td>64 (60.4)</td>
<td>98 (62.0)</td>
</tr>
<tr>
<td>Women</td>
<td>18 (34.6)</td>
<td>42 (39.6)</td>
<td>60 (38.0)</td>
</tr>
<tr>
<td>Duration of AD, y, mean (SD)</td>
<td>24.8 (11.1)</td>
<td>24.7 (9.7)</td>
<td>24.8 (10.2)</td>
</tr>
<tr>
<td>mEASI score, mean (SD)</td>
<td>14.5 (3.8)</td>
<td>14.2 (3.5)</td>
<td>14.3 (3.6)</td>
</tr>
<tr>
<td>IGA score, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 (moderate)</td>
<td>36 (69.2)</td>
<td>73 (68.9)</td>
<td>109 (69.0)</td>
</tr>
<tr>
<td>4 (severe)</td>
<td>16 (30.8)</td>
<td>33 (31.1)</td>
<td>49 (31.0)</td>
</tr>
<tr>
<td>Face/neck IGA score, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 (clear)</td>
<td>2 (3.8)</td>
<td>5 (4.7)</td>
<td>7 (4.4)</td>
</tr>
<tr>
<td>1 (almost clear)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2 (mild)</td>
<td>7 (13.5)</td>
<td>10 (9.4)</td>
<td>17 (10.8)</td>
</tr>
<tr>
<td>3 (moderate)</td>
<td>28 (53.8)</td>
<td>64 (60.4)</td>
<td>92 (58.2)</td>
</tr>
<tr>
<td>4 (severe)</td>
<td>15 (28.8)</td>
<td>27 (25.5)</td>
<td>42 (26.6)</td>
</tr>
<tr>
<td>Pruritus NRS score, mean (SD)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime score</td>
<td>5.4 (2.3)</td>
<td>5.3 (2.1)</td>
<td>5.3 (2.2)</td>
</tr>
<tr>
<td>Nighttime score</td>
<td>4.8 (2.4)</td>
<td>4.6 (2.4)</td>
<td>4.6 (2.4)</td>
</tr>
<tr>
<td>Percentage of BSA affected by AD, mean (SD)</td>
<td>23.0 (5.2)</td>
<td>23.5 (5.3)</td>
<td>23.3 (5.2)</td>
</tr>
</tbody>
</table>

AD, Atopic dermatitis; BSA, body surface area; IGA, Investigator’s Global Assessment; mEASI, modified Eczema Area and Severity Index; NRS, numeric rating scale; SD, standard deviation.

Efficacy

In part 1, the least-squares mean percent changes from baseline in mEASI score were -44.3% in the delgocitinib group and 1.7% in the vehicle group at EOT (Fig 1). mEASI score in the delgocitinib group was significantly reduced compared with that in the vehicle group (P < .001). mEASI score in the delgocitinib group continued to be reduced from week 1 through week 4. Similarly, the other efficacy parameters at EOT, such as IGA and pruritus NRS scores, were significantly improved in the delgocitinib group compared with those in the vehicle group (Supplemental Tables I and II; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

More patients in the delgocitinib group achieved mEASI-50 and mEASI-75 at EOT than in the vehicle group (Fig 2). The proportion of patients with a mEASI-50 was 51.9% (55 of 106) in the delgocitinib group and 11.5% (6 of 52) in the vehicle group (P < .001). The proportion of patients with a mEASI-75 was 26.4% (28 of 106) in the delgocitinib group and 5.8% (3 of 52) in the vehicle group (P < .01). Similarly, IGA response rates at EOT in the delgocitinib group were higher (not significant for the overall score) than in the vehicle group (P = .32 for the overall score, P < .05 for the face/neck score) (Supplemental Fig 3; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

The pruritus NRS score in the delgocitinib group was lower than in the vehicle group at week 1, which was maintained over time (Supplemental Fig 4; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1). The daily changes in the score
showed a rapid reduction in pruritus after the start of study treatment in the delgocitinib group (Fig 3).

Long-term treatment with delgocitinib maintained the improvement in the mEASI, IGA, pruritus NRS scores (Supplemental Table III; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1), and the proportion of patients with mEASI-50 and mEASI-75 (Supplemental Fig 5; available via
noted in part 1. At week 24, the mean percent change from baseline in the mEASI score was -56.3%, and the proportions of patients with mEASI-50 and mEASI-75 were 69.3% (95 of 137) and 35.8% (49 of 137), respectively.

Representative photographs of patients treated with delgocitinib show improvement in signs of AD and support the efficacy results (Supplemental Fig 6; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

Safety and tolerability

In part 1, adverse events (AEs) were reported in 23 of 106 (21.7%) patients in the delgocitinib group and in 6 of 52 (11.5%) patients in the vehicle group (Supplemental Table IV; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1). No serious AEs, severe AEs, or AEs leading to study discontinuation were reported. The majority of AEs were considered mild. Treatment-related AEs were reported in 5 of 106 (4.7%) patients in the delgocitinib group and 1 of 52 (1.9%) patients in the vehicle group.

Across part 1 and part 2, AEs were reported in 78 of 154 (50.6%) patients after the start of delgocitinib treatment (Table II). No serious or severe AEs were reported. The majority of AEs were considered mild. Study discontinuations due to AEs occurred in only 1 patient. The most common AE was nasopharyngitis (n = 30 [19.5%]), followed by Kaposi varicelliform eruption (n = 6 [3.9%]) and acne (n = 5 [3.2%]). No irritation symptoms, such as application site burning, stinging, or redness, were found. Treatment-related

| Table II. Summary of adverse events over the treatment period with delgocitinib 0.5% ointment for up to 28 weeks* |
|-----------------------------------------------|-----------------|
| Adverse events                              | Total (N = 154) |
| Adverse events                              | 78 (50.6)       |
| Maximum severity                            |                 |
| Mild                                         | 68 (44.2)       |
| Moderate                                     | 10 (6.5)        |
| Severe                                       | 0               |
| Treatment-related adverse events             | 9 (5.8)         |
| Serious adverse events                       | 0               |
| Adverse events leading to discontinuation    | 1 (0.6)         |
| Adverse events occurring in ≥2% of patients  |                 |
| Nasopharyngitis                              | 30 (19.5)       |
| Kaposi varicelliform eruption                | 6 (3.9)         |
| Acne                                         | 5 (3.2)         |
| Dental caries                                | 4 (2.6)         |
| Paronychia                                   | 4 (2.6)         |
| Pyrexia                                      | 4 (2.6)         |
| Treatment-related adverse events occurring in ≥1% of patients | 3 (1.9)       |

*Data are displayed as number of patients (%). No data from part 1 in the vehicle group are included; thus, patients with adverse events during delgocitinib treatment are counted.

Fig 3. Daily change (least-squares mean and 95% confidence interval) from baseline in pruritus numeric rating scale (NRS) score over the first week of treatment. D, daytime; N, nighttime. *P < .01, **P < .001 versus vehicle.
AEs were reported in 9 of 154 (15.4%) patients; the most common treatment-related AE was Kaposi varicelliform eruption (n = 3 [1.9%]). The incidence of AEs did not increase over time (Supplemental Table V; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

**Pharmacokinetics**

No plasma concentrations of delgocitinib were detected in most patients during the study (85.5%-91.1%). No apparent difference between study visits was found in the proportion of patients with detectable plasma concentrations of delgocitinib. The maximum plasma concentration of delgocitinib at each study visit ranged from 4.3 ng/mL to 11.4 ng/mL (Supplemental Table VI; available via Mendeley at https://doi.org/10.17632/tpx4fwkjny.1).

**DISCUSSION**

In the present study, delgocitinib 0.5% ointment rapidly improved clinical signs and symptoms in Japanese adult patients with moderate to severe AD. The improvement effect on AD was maintained for up to 28 weeks, and long-term treatment with delgocitinib 0.5% ointment was well tolerated. Efficacy and safety results in AD were also obtained in recent clinical studies of other JAK inhibitors, such as tofacitinib,41 ruxolitinib,55 baricitinib,56 and ASN002.37,38 The present study provides additional evidence that topical JAK inhibitors are a promising therapeutic option for AD.

Biologics inhibiting cytokine signaling have been found to be effective in the treatment of AD. Dupilumab, an inhibitor of both IL-4 and IL-13 signaling, is used in clinical settings.39,40 Nemolizumab, an inhibitor of IL-31 signaling, has received attention for its antipruritic effect.41,42 As with other cytokines, IL-4, IL-13, and IL-31 exert their biological effects via the JAK-STAT pathway. Although the route of administration is different (systemic vs topical), the clinical evidence of both biologics can support the potential efficacy of delgocitinib ointment. Additionally, delgocitinib, a pan-JAK inhibitor, can broadly inhibit other cytokine signaling, which is considered a better profile, given that many cytokines are involved in the pathophysiology of AD.

Pruritus is a distressing symptom in patients with AD, leading to impairments of quality of life such as sleep disturbance, increased risk of secondary infection, and further exacerbation of AD due to scratching.53 As shown in the phase 2 study,30 delgocitinib ointment rapidly reduced the pruritus NRS score in the present study. This antipruritic effect of delgocitinib ointment can assist in reducing distress in patients with AD.

Overall, delgocitinib ointment was well tolerated over the treatment period. The majority of AEs were mild and unrelated to delgocitinib. In part 1 (4-week double-blind period), however, the incidence of AEs was generally low but was higher in the delgocitinib group (21.7%) than in the vehicle group (11.5%). This difference may be attributable to a shorter assessment period in the vehicle group because more patients in the vehicle group (38.5%) entered part 2 (24-week extension period) early without completing part 1 than those in the delgocitinib group (7.5%) owing to worsening of AD. In fact, the mean duration of exposure to study treatment was shorter in the vehicle group (20.7 days) than in the delgocitinib group (27.8 days). In the phase 2 study,30 no apparent difference in the incidence of AEs was found between the delgocitinib 0.5% ointment (18.5%) and vehicle (15.6%) groups.

At the application sites of delgocitinib ointment, no skin atrophy or telangiectasia, as seen with long-term use of topical corticosteroids,5 was found. No irritation symptoms such as burning sensations, as commonly reported with tacrolimus ointment,5 were found. Local skin infections, including Kaposi varicelliform eruption, were infrequent; however, appropriate monitoring of local skin infections is mandatory given the immune-suppressive activity of delgocitinib. Systemic exposure to delgocitinib was low throughout the treatment period; thus, delgocitinib ointment is unlikely to cause systemic infections due to excessive immunosuppression. Overall, the safety results suggest that delgocitinib ointment has a favorable safety profile as a topical drug for AD.

The present study has some limitations. First, because only Japanese patients were included, it is unclear whether the study results are applicable to non-Japanese patients who have different clinical phenotypes of AD.9,10 Delgocitinib ointment, however, targets multiple cytokine axes and is potentially effective in those populations. Second, part 2 was conducted in an open-label manner without any control group, which may have led to assessment bias. Third, the vehicle-controlled period lasted only 4 weeks. Finally, in part 2, concomitant use of topical corticosteroids was allowed for the treatment of worsening of AD. These last 2 factors limit discussions on the long-term efficacy of delgocitinib treatment.

In conclusion, delgocitinib 0.5% ointment was effective and well tolerated in Japanese adult patients with moderate to severe AD for up to 28 weeks. The study results indicate that delgocitinib ointment is a promising therapeutic option for AD.

We would like to thank the patients who participated in the study, as well as the investigators and staff at the study sites (Supplemental Table VII; available via Mendeley at
REFERENCES


